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<u>L1</u>	5328984	2	<u>L1</u>

END OF SEARCH HISTORY

=> d 14 1-3 bib ab

L4 ANSWER 1 OF 3 IFIPAT COPYRIGHT 2001 IFI DUPLICATE 1
AN 3582569 IFIPAT;IFIUDB;IFICDB
TI MUTAGENIZED IL 13-BASED CHIMERIC MOLECULES
INF Debinski; Waldemar, Hershey, PA
IN Debinski Waldemar
PAF The Penn State Research Foundation
PA Penn State Research Foundation The (31470)
EXNAM Eyler, Yvonne
EXNAM Andres, Janet L
AG Senterfitt, Akerman
PI US 6296843 20011002
AI US 1998-54711 19980403
FI US 6296843 20011002
DT UTILITY
FS CHEMICAL
CLMN 12
GI 6 Drawing Sheet(s), 11 Figure(s).
AB This invention provides mutagenized interleukin 13 molecules that show improved specificity for the restricted (IL4 independent) IL13 receptor and reduced cross-reactivity with the IL4/IL4 shared receptor. The mutagenized IL13 molecules include one or more mutations in a domain that interacts with the 140 kDa hIL4R beta or the hIL13R alpha1 subunit. These mutagenized IL13 molecules provide effective targeting moieties in chimeric molecules (e.g. fusion proteins) that specifically deliver effector molecules (e.g. cytotoxins) to cells overexpressing IL13 receptors (e.g. cancer cells such as gliomas).

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 2
AN 1999:659425 CAPLUS
DN 131:285412
TI Mutagenized IL13-based chimeric molecules
IN Debinski, Waldemar
PA The Penn State Research Foundation, USA
SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9951643	A1	19991014	WO 1999-US7188	19990331
	W:				
	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ,				
TM					
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6296843	B1	20011002	US 1998-54711	19980403
	AU 9933774	A1	19991025	AU 1999-33774	19990331
	EP 1071717	A1	20010131	EP 1999-915196	19990331

R: AT, CH, DE, ES, FR, GB, IT, LI, SE

PRAI US 1998-54711 A 19980403

WO 1999-US7188 W 19990331

AB This invention provides mutagenized interleukin 13 mols. that show improved specificity for the restricted (**IL4** independent) **IL13 receptor** and reduced cross reactivity with the **IL4/IL4** shared receptor. The mutagenized **IL13** mols. include one or more mutations in a domain that interacts with the 140 kDa h**IL4**R.beta. or the h**IL13**R.alpha.1 subunit. These mutagenized **IL13** mols. provide effective targeting moieties in chimeric mols. (e.g. fusion proteins) that specifically deliver effector mols. (e.g. cytotoxins) to cells overexpressing **IL13** receptors (e.g. cancer cells such as gliomas).

RE.CNT 5

RE

(1) Debinsky, W; Nature Biotechnology 1998, V16, P449

(2) Debinsky, W; The Journal of Biological Chemistry 1995, V270(28), P16775

(3) Maini, A; The Journal of Urology 1997, V158, P948 CAPLUS

(4) Penn State Research Foundation; WO 98/19857 A1 1998 CAPLUS

(5) The Government of the United States of America; WO 96/29417 A1 1996 CAPLUS

L4 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 3

AN 1998:273691 BIOSIS

DN PREV199800273691

TI Novel way to increase targeting specificity to a human glioblastoma-associated receptor for interleukin 13.

AU Debinski, Waldemar (1); Gibo, Denie M.; Puri, Raj K.

CS (1) Sect. Neurosurgery, H110, Dep. Surg., Milton S. Hershey Med. Cent., Pa. State Univ. Coll. Med., 500 University Drive, Biomedical Res. Building, C3848A, Hershey, PA 17033-0850 USA

SO International Journal of Cancer, (May 18, 1998) Vol. 76, No. 4, pp. 547-551.

ISSN: 0020-7136.

DT Article

LA English

AB Human brain cancers (gliomas) overexpress large numbers of a receptor for interleukin 13 (**IL13**), making this receptor an attractive target for anti-glioma therapies. We have recently proposed that the glioma-associated **IL13 receptor** is different from the one expressed on some hemopoietic and somatic cells. In an attempt to identify an even more glioma-specific target, we have used an antagonist of a related cytokine, **IL4**, which neutralizes the physiological effects

of

both **IL13** and **IL4** on normal cells. Here we demonstrate that the **IL4** antagonist also counteracts the action of cytotoxins targeted to the **IL13 receptor** on normal human cells. Importantly, the **IL4** antagonist does not inhibit **IL13**-based cytotoxins on glioma cells at all. Thus, the **IL13 receptor** on glioma cells can be categorized as tumor-specific in the presence of an **IL4** antagonist. We conclude that **IL13 receptor**-directed cytotoxins can be delivered to glioma cells without being cytotoxic to normal cells.

(FILE 'HOME' ENTERED AT 15:42:10 ON 26 APR 2002)

INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 15:42:40 ON 26 APR 2002

SEA GLIOMA(25W) (TREAT? OR ADMIN?) AND INTRATUM?

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1 FILE BIOBUSINESS
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59 FILE MEDLINE
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5 FILE PROMT
40 FILE SCISEARCH
35 FILE TOXCENTER
30 FILE USPATFULL
1 FILE WPIDS
1 FILE WPINDEX

L1 QUE GLIOMA(25W) (TREAT? OR ADMIN?) AND INTRATUM?

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L2 531 S GLIOMA(25W) (TREAT? OR ADMIN?) AND INTRATUM?
L3 191 S L2 AND (TREAT? OR ADMIN?) (15W) INTRATUM?
L4 80 DUP REM L3 (111 DUPLICATES REMOVED)

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d his

(FILE 'HOME' ENTERED AT 12:31:42 ON 16 MAY 2000)

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, AIDSLINE, ANABSTR, AQUASCI,
BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO,
CABA,
CANCERLIT, CAPLUS, CƏABA, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU,
DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 12:31:52 ON 16 MAY
2000

SEA GLIOBLASTOMA AND 251 GLIOMA

SEA GLIOBLASTOMA AND 251 MG GLIOMA

1 FILE CAPLUS

L1 QUE GLIOBLASTOMA AND 251 MG GLIOMA

FILE 'CAPLUS' ENTERED AT 12:33:31 ON 16 MAY 2000

L2 1 S GLIOBLASTOMA AND 251 MG GLIOMA

(FILE 'HOME' ENTERED AT 13:12:35 ON 16 MAY 2000)

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, AIDSLINE, ANABSTR, AQUASCI,
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CABA,
CANCERLIT, CAPLUS, CEABA, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU,
DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 13:12:51 ON 16 MAY
2000

SEA 251 MG AND IL-13

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2 FILE CAPLUS
2 FILE EMBASE
1 FILE ESBIODASE
1 FILE LIFESCI
2 FILE MEDLINE
1 FILE PROMT
2 FILE SCISEARCH
1 FILE TOXLIT

L1 QUE 251 MG AND IL-13

FILE 'CANCERLIT, BIOTECHNO, CAPLUS, EMBASE, MEDLINE, SCISEARCH, BIOSIS,
ESBIODASE, LIFESCI, PROMT, TOXLIT' ENTERED AT 13:15:05 ON 16 MAY 2000

L2 18 S 251 MG AND IL-13

L3 4 DUP REM L2 (14 DUPLICATES REMOVED)

FILE 'USPATFULL' ENTERED AT 13:21:23 ON 16 MAY 2000

L4 59 S INTRATUMORAL INJECTION

BEST AVAILABLE COPY

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS
AN 1993:188930 CAPLUS
DN 118:188930
TI The presence of neuron-associated microtubule proteins in the human U-251
MG cell line: a comparative immunoblot and immunohistochemical study
AU Lopes, M. Beatriz S.; Frankfurter, Anthony; Zientek, Gary M.; Herman,
Mary
M.
CS Dep. Pathol., Univ. Virginia, Charlottesville, VA, USA
SO Mol. Chem. Neuropathol. (1992), 17(3), 273-87
CODEN: MCHNEM; ISSN: 1044-7393
DT Journal
LA English
AB U-251 MG, a permanent cell line derived from human **glioblastoma**
multiforme with the capacity to maintain glial fibrillary acidic protein
(GFAP) prodn. over repeated in vitro passages, was evaluated for the
expression of 3 neuron-assocd. proteins (class III .beta.-tubulin, MAP2,
and tau) in 3 different in vitro systems: as free-floating suspensions,
on coverslips, and on a gelatin foam (Gelfoam) matrix. Cells grown under
the 3 in vitro conditions were analyzed by immunoblotting techniques, whereas
immunohistochem. analyses were performed on cells grown on Gelfoam. By
immunohistochem., cells were pos. for class III B-tubulin isotype, a
neuron-assocd. B-tubulin, for MAP2, but not for tau. Immunoblotting
studies confirmed the presence of class III .beta.-tubulin in exts. of
cells grown under the 3 in vitro conditions. MAP2 and tau were clearly
evident only in cell exts. grown in Gelfoam cultures. GFAP expression
was obsd. in all 3 in vitro conditions by immunoblotting and also in foam
matrix cultures by immunohistochem. In matrix cultures, class III
.beta.-tubulin- and GFAP-pos. cells were found immediately adjacent to
each other, but co-expression of these proteins was not obsd., and the
cells were morphol. indistinguishable. The findings confirm the
heterogeneity of malignant gliomas in vitro, and the implications of
these observations require further study.

L3 ANSWER 2 OF 4 CANCERLIT
AN 96394300 CANCERLIT
DN 96394300

DUPLICATE 1

TI Receptor for interleukin (IL) 13 does not interact
with IL4 but receptor for IL4 interacts with IL13 on human glioma cells.
AU Debinski W; Miner R; Leland P; Obiri N I; Puri R K
CS Department of Surgery, The Milton S. Hershey Medical Center, Pennsylvania
State University, Hershey, Pennsylvania 17033-0850, USA.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1996). Vol. 271, No. 37, pp. 22428-33.
Journal code: HIV. ISSN: 0021-9258.

DT Journal; Article; (JOURNAL ARTICLE)
FS MEDL; L; Priority Journals; Cancer Journals

LA English
OS MEDLINE 96394300
EM 199612

AB Recently, we have demonstrated that human (h) glioma cell lines express
large number of receptors (R) for interleukin 13 (IL13) (Debinski, W.,
Obiri, N. I., Powers, S. K., Pastan, I., and Puri, R. K. (1995) Clin.
Cancer Res. 1, 1253-1258). These cells are extremely sensitive to a
chimeric protein composed of hIL13 and a derivative of Pseudomonas
exotoxin (PE), PE38QQR. We have found that the cytotoxicity of
hIL13-PE38QQR was blocked by hIL13 but not by hIL4 on the U-251
MG and U-373 MG cells, contrary to what was observed on several
adenocarcinoma cell lines. In the present study, we further explored
interactions between receptor for IL13 and IL4 on glioma cells.
Established human glioma cell lines, such as DBTRG MG, Hs 683, U-87 MG,
SNB-19, and A-172, are very susceptible to hIL13-PE38QQR, and the action
of the chimeric toxin is not blocked by hIL4 on all these cells either.
Also, hIL4 is not a competitor for 125I-hIL13 binding sites on glioma
cells. Of interest, a corresponding hIL4-based chimeric toxin,
hIL4-PE38QQR, is poorly active or not active on all the tested glioma

cell lines. When active, however, hIL4 toxin action was blocked by hIL13.

hIL13 is a competitor for 125I-hIL14 binding in a competitive binding assay on
glioma cells. hIL13 and hIL4 did not affect the growth of the tested
glioma cell lines. Human glioblastoma multiforme explant cells exhibited
similar responses to the chimeric toxins and interleukins when compared
with that found in established glioma cultures. Our results suggest that
the hIL13R on glioma cells is expressed in one predominant form, the form
that does not interact with IL4. Thus, this type of hIL13R is apparently
different from the one demonstrated previously on several adenocarcinoma
cell lines.

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(FILE 'HOME' ENTERED AT 11:59:45 ON 30 OCT 2001)

INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
CABA, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO,
CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 12:04:19 ON
30 OCT 2001

SEA GLIOBLASTOMA MULTIFORME(15W) (BRAIN OR CRANIUM)

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7 FILE BIOCOMMERCE
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117 FILE SCISEARCH
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2 FILE WPIDS
2 FILE WPINDEX

L1 QUE GLIOBLASTOMA MULTIFORME(15W) (BRAIN OR CRANIUM)

SEA GLIOBLASTOMA MULTIFORME(10W) (BRAIN OR CRANIUM)

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2 FILE ADISINSIGHT
4 FILE ADISNEWS

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 61 FILE PASCAL
 1 FILE PHAR
 15 FILE PHIN
 87 FILE PROMT
 102 FILE SCISEARCH
 19 FILE TOXLIT
 17 FILE USPATFULL
 2 FILE WPIDS
 2 FILE WPINDEX

L2 QUE GLIOBLASTOMA MULTIFORME(10W) (BRAIN OR CRANIUM)

 FILE 'CANCERLIT, MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS, PROMT,
 PASCAL, ESBIODASE, BIOTECHNO, LIFESCI, TOXLIT, USPATFULL, DRUGU, PHIN,
 JICST-EPLUS, CIN, DGENE, BIOCOMMERCE, ADISNEWS, ADISALERTS, BIOTECHDS,
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 30 OCT 2001

FILE 'CANCERLIT, MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS, PROMT,
 PASCAL, ESBIODASE, BIOTECHNO, LIFESCI, TOXLIT, USPATFULL, DRUGU, PHIN,
 JICST-EPLUS, CIN, DGENE, BIOCOMMERCE, ADISNEWS, ADISALERTS, BIOTECHDS,
 ADISINSIGHT, BIOBUSINESS, CEABA-VTB, DRUGNL, ...' ENTERED AT 12:11:32 ON
 30 OCT 2001

L3 0 S GLIOBLASTOMA MULTIFORME(10W) (CRANIUM)
 L4 1139 S GLIOBLASTOMA MULTIFORME(10W) (BRAIN)
 L5 449 DUP REM L4 (690 DUPLICATES REMOVED)

L4 ANSWER 66 OF 80 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 1987:128041 BIOSIS

DN BA83:67102

TI EXPERIMENTAL AND CLINICAL STUDIES ON **INTRATUMORAL** CHEMOTHERAPY
IN SUPRATENTORIAL MALIGNANT GLIOMAS.

AU SASAHIRA M

CS DEP. OF NEUROSURGERY, FAC. OF MED., KAGOSHIMA UNIV., KAGOSHIMA, 890, JPN.

SO MED J KAGOSHIMA UNIV, (1986) 38 (1), 15-48.

CODEN: KDIZAA. ISSN: 0368-5063.

FS BA; OLD

LA Japanese

AB Recently, many therapeutic procedures for malignant brain tumors have been developed and the results of these have been much improved. According to the many reports on brain tumors chemotherapy, it was well not always satisfactory, therefore, the authors performed the **intratumoral** injection in which high concentration of antitumor drugs remain for a long time in the tumor cavity. In this paper, as a part of our serial investigation on the brain tumor chemotherapy, the author has investigated the effects of **intratumoral** chemotherapy and the following histological changes of transplanted tumors and clinical cases I. Experimental studies: The effects of anti-tumor drugs (methotrexate, betamethasone, ACNU and OK-432) on the rat brain tumor models were investigated from the viewpoints of survival time and histological findings. Followings are the materials and methods employed in this experiment. A rat **glioma** cell line (RG12 cell line) originally induced in CDF rat by **administration** of nitrosourea and has been maintained with tissue culture. The tumor cells suspension, 1 .times. 105 cells/0.01 ml, was injected into the frontal lobe in the CDF rats. Rats were grouped into four experimental designations. One was a control group and the others were administered groups. They were ACNU group. betamethasone (abbreviated to BMS) with methotrexate (abbreviated to MTX) group and OK-432 (abbreviated to OK) group. The drugs were administered at a dose of 0.025 K.E. in OK group, 0.5 mg in ACNU group and 0.2 mg of MTX with 0.2 mg of BMS in MTX with BMS group, 7 days, 10 days and 16 days after inoculation. The results were as follows: 1) Mean survival days; MTX with BMS group was 85.8 .+- 31.8 days, ACNU group was 56.6 .+- 33.9 days, OK group was 53.8 .+- 34.3 days and control group was 42.3 .+- 7.0 days. 2) Histological findings showed that the necrotic areas in the tumor spreaded much wider in the drugs administered group than the control group. Infiltration of lymphocytic cells were observed in the peripheral areas of the tumors. This phenomenon was more prominent in the OK group. II. Clinical studies: The clinical application of **intratumoral** chemotherapy was performed on 32 cases of supratentorial gliomas. The antitumor drugs (MTX + BMS, BMS only, ACNU + BMS and MTX + BMS + ACNU) were **administered** during the periods of radiation therapy. The results were as follows: 1) Survival time; **Intratumoral** chemotherapy group low grade astrocytoma (abbreviated to A-I group): 7-86 month glioblastoma (abbreviated to A-II group): 3-73 month Non-**intratumoral** chemotherapy group low grade astrocytoma (abbreviated to B-I group): 11-88 month glioblastoma (abbreviated to B-II group): 5-40 month Mean survival time; A-I 36.6 mont, A-II 21.5 month B-I 48.8 month, B-II 19.2 month 2) Survival rates; A-I; 1 year 86.7%, 2 year 73.3%, 3 year, 50.0% 4 year 50.0% 5 year 40.0% A-II; 1 year 63.2%, 2 year 37.9%, 3 year 31.6% 4 year 21.1%, 5 year 21.1% B-I; 1 year 94.1%, 2 year 94.1%, 3 year 82.4% 4 year 75.4%, 5 year 66.1% B-II; 1 year 50.0%, 2 year 50.0%, 3 year 15.0% 4 year 0%, 5 year 0% 3) Improvement of performance state after the therapy A-I; CR 68.7%, PR 12.5%, ST 18.8%, PG 0% A-II; CR 57.9%, PR 21.1%, ST 21.1%, PG 0% B-I; CR 64.7%, PR 23.5%, ST 11.8%, PG 0% B-II; CR 40.0%, PR 40.0%, ST 20.0%, PG 0% 4) Reduction rate of tumor volume after the therapy A-I; CR 43.8%, PR 25.0%, ST 31.3%, PG 0% A-II; CR 15.8%, PR 26.3%, ST 52.6%, PG 5.3% B-I; CR 40.0%, PR 40.0%, ST 13.3%, PG 6.7% B-II; CR 14.3%, PR 71.4%, ST 14.3%, PG 0% 5) Improvement of angiographical findings after the therapy Improvement of mass effects were well noticed

in each group. Disappearance of tumor stains were as followed. (A-I; 50.0%, A-II; 60.0%, B-I; 100%, B-II; 100%) 6) The reduction rate of tumor volume keeps a regular relation to the removal rate of tumor and the case, the implanter is setted the middle portion of tumor, is more effective than the case whose implanter is setted the periphery of tumor. 7) Histological findings Coagulation necrosis of tumor was found near the implanter, but proliferation of tumor cells were found at the remote area from the implanter. 8) Side effects of **intratumoral** chemotherapy There was no complication of central nervous system. The infection only is one of complications. The purpose of this **intratumoral** chemotherapy is to by-pass the blood-brain barrier and to expose the neoplasmtic tissue directly to the drugs. In addition, high concentration of the drugs could be achieved with a very low passage of the compounds into the blood stream, therefore, considerably reducing the systemic toxicity. The **intratumoral** chemotherapy will be useful one and should be carried out for malignant brain tumors not only as induction therapy but also as maintenance therapy.

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